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ABSTRACT

A clinical and genetic analysis was made on the basis of 52 families: 27 were from reading clinics where at least one child in the family had been diagnosed as having a reading disability; 25 were families of children selected from a third-, fourth-, or fifth-grade class on the basis of having average or above-average IQ and achievement. Through the use of a questionnaire a summary was made of specific reading disability characteristics found in the families and the students. It was found that a larger number of males than females was affected with a reading disability. Birth order did not appear to be a contributing factor. Only one family was present where both parents had a reading disability; both of their children were affected with a reading disability. In the families where one parent was affected with a reading disability, Weinberg's method of Mendelian Analysis was used, giving a value of 37.7 percent of their children being affected. Chi-square analysis indicated that the value of 37.7 percent is not significantly different from the theoretical 50 percent expected in a single hybrid autosomal dominant mode of inheritance. Thus it was concluded that a relationship between heredity and specific reading disability was shown. Other biochemical findings, tables, and a bibliography are included. (Author/DH)

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THE ROLE OF HEREDITY IN READING DISABILITY

U. S. DEPARTMENT OF HEALTH, EDUCATION & WELFARE
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by

Laryl Lee Delker

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Chapter 1

INTRODUCTION

It has been estimated that eight million children in American elementary and secondary schools do not learn to read adequately. That is, about one child in seven, or 15 percent of the total school population, is handicapped in his ability to master the skills necessary to read.[1] Below average intelligence, brain damage, illness, poor eyesight, partial hearing, immaturity, lack of interest, emotional problems, poor social-economic background and poor teaching are listed as possible causes for this failure. Although these conditions may often cause reading failure, there is another cause often overlooked that may play an important role in the diagnosis of reading problems, the role heredity plays in the reading problem.

In the group of poor readers is an unrecognized minority of children who have what is called specific reading disability. These children are unable to compete with their classmates in reading, writing and spelling in

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[1]U. S. Government Department of Health, Education and Welfare, Reading Disorders in the United States, Report of the Secretary's (H. E. W.) National Advisory Committee on Dyslexia and Related Reading Disorders (Bethesda, Maryland: National Institute of Health, 1969), pp. 21-23.

the normal school situation. They are intelligent, healthy children from good homes with little evidence of neurological damage. They try hard, but fail to learn to read. Some of these children seem to have a right and left confusion for a longer time than is normal. They find it hard to remember the shapes of different letters and words on the printed page, as well as the sound of some letters. Since specific reading disability is not felt to be caused by injury, disease, emotional problems or any other identifiable cause,[2] it may be that these children are born lacking the ability to do certain things which are either necessary for reading or must be compensated for before success in reading can result.

Since 1905, when Thomas and Fisher first reported cases of "Congenital Word Blindness,"[3] much has been reported in the literature to suggest a genetic factor in some cases of specific reading disability. Most writers have published descriptive reports of families having specific reading disability in several generations. Hallgren (1950) on the basis of 116 cases of specific reading disability in a study of 276 families was able to conclude:

> The genetic statistical analysis shows that specific dyslexia (specific reading disability)

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[2]Bertil Hallgren, "Specific Dyslexia ('Congenital Word Blindness') A Clinical and Genetic Study," Acta Psychiatrica et Neurologica, Suppl., LXV, 1 (1950), 226.

[3]J. Hinshelwood, Congenital Word-Blindness (London: H. K. Lewis and Co., Ltd., 1917), p. 40.

with a high degree of probability, follows a monohybrid autosomal dominant genetic tendency.[4]

Thus, a single gene may be involved in a dominant non-sex linked form of inheritance. Hallgren's work is the most complete study available. In the last twenty years smaller studies have been undertaken, but they also use a personal interview to obtain family background information.

It is quite difficult to obtain trustworthy family background by interviewing one family member. Usually only the extreme instances of a disability of other family members can be noted or remembered. Therefore a study of the inheritance of specific reading disability was needed in which information was gathered other than by personal interview. In this study, a questionnaire, called the Family Check List, was sent to the home to be filled out. This attempted to provide better conditions for getting more reliable answers to questions relating to reading disorders.

THE PROBLEM

Statement of the Problem

The purpose of this study was to investigate the relationship between heredity and specific reading disability. From the review of the research, a questionnaire was developed to obtain family genetical history that is related to specific reading disability. This questionnaire

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[4]Hallgren, op. cit., pp. 231-232.

was designed for use by reading specialists as another tool in diagnosing the child with a reading problem.

Null Hypothesis

There is no significant relationship between heredity and specific reading disabilities.

Importance of the Study

Reading specialists have been impressed with the regularity with which parents and relatives of children with specific reading disabilities tell of other family members having a similar problem.[5] Until the last few years psychologists thought the conditions of "word-blindness" and related reading problems were the result of a non-specific reaction to a series of environmental factors. Now, the same psychologists have moved toward the current medical opinion that part of the large number of children with reading handicaps may suffer from a congenital form of reading disability.[6]

The part that heredity plays in reading problems has received the least investigation of all the recognized possible causes of reading disabilities. It is not a clear cut problem that can be identified as easily as color

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[5]Leon Eisenberg, "The Epidemiology of Reading Retardation and a Program for Preventive Intervention," The Disabled Reader, ed. J. Money (Baltimore: The Johns Hopkins Press, 1966), p. 16.

[6]Knud Herman, Reading Disability (Springfield, Ill.: C. C. Thomas, 1959), p. 10.

blindness or deafness. No questionnaire, check list, schedule or form appears in the literature that can be used to obtain family background information relevant to genetic characteristics and reading disability. Questionnaires are available to obtain the information on a child's pre-natal to present age development. Neurological, psychological, school and medical forms are also available. Therefore, a questionnaire that could be understood and used by a reading teacher needed to be developed and evaluated. Thus, the present researcher devised a questionnaire as an added tool that could be used in the diagnosis of reading problems.

Limitations of the Study

1. The most complete primary genetic study available is Hallgren's "A Clinical and Genetic Study of Specific Dyslexia" published in Stockholm in 1950.[7] Hallgren's study was used as a basis for the present study.

2. The results of the questionnaire are only as valid as the questions asked.

3. The questionnaire was distributed to parents to complete at home. The data collected were limited by the truthfulness and memory of the parents.

4. The questionnaire was given to three test groups:

A. Families of children diagnosed as having specific reading disability at the University

[7]Hallgren, op. cit.

of Pennsylvania Reading Clinic. Sample size of this population was limited to four.

B. Families of children who came to the Glassboro Reading Clinic because of reading problems. The selection of these cases was limited by the judgment of the researcher as being a specific reading disability subject. The sample size of this population was limited to twenty-four.

C. Families of children who are reading at their expected reading level, having an average or better intelligence, and experiencing little difficulty in reading. These children were randomly selected by the researcher. The size of this population was limited to twenty-five.

5. The depth of this study was limited because of a short time factor. More extensive testing may be necessary before the validity of the questionnaire can be established.

DEFINITIONS OF TERMS USED

Environment

Environment refers to the sum of the external conditions and factors potentially capable of influencing the organism.[8]

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[8]Horace B. English and A. C. English, A Compre-

Family

In this study the family refers to the student (subject studied), siblings, parents, paternal/maternal grandparents. All of the research sample families were investigated through and inclusive of the third generations. Adopted relatives or relatives not related by blood were eliminated from this study.

Genetics

Genetics is the science of heredity.[9]

Heredity

The totality of influences, biologically transmitted from parents that determine the ways in which an individual will make use of his environment is referred to as heredity.[10]

Proband

Proband refers to affected individual being studied.[11]

Specific Reading Disability

"Also known as congenital word blindness (Morgan, 1896), primary reading retardation (Rabinovitch, et. al.,

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hensive Dictionary of Psychological and Psychoanalytical Terms (New York: David McKay Co., 1965).

[9]Ibid.

[10]Ibid.

[11]Curt Stern, Principles of Human Genetics (San Francisco: W. H. Freeman dna Co., 1960), p. 134.

1954) and developmental dyslexia (Critchley, 1964)." The adjective 'specific' calls attention both to the circumscribed nature of the disability and to our ignorance of its cause. Operationally, it may be defined as a failure to learn to read with normal proficiency despite conventional instruction, a culturally adequate home, proper motivation, intact senses, normal intelligence, and freedom from gross neurological defects.[12]

[12]Eisenberg, op. cit., p. 14.

Chapter 2

REVIEW OF THE LITERATURE

Reading disabilities, unless associated with obvious and gross cerebral damage, have come to be regarded as a psychological or pedagogical problem apparently unrelated to the functions of the central nervous system. Some writers on the subject have concluded that there is little evidence of organic or inherited neurological dysfunction in cases of specific reading disability in the intelligent child.[1] It is part of the purpose of this thesis to examine the most widely quoted papers that have been published to see what support there is for the hypothesis that specific reading disability is inherited.

In reviewing the literature, it is noted that this reading difficulty has been called by many names. It was first called "congenital word-blindness" by Morgan (1896) when it was introduced as a clinical entity. Since that time a great many other terms have been suggested and used. Among them are strephosymbolia (Orton, 1928), specific dyslexia (Hallgren, 1950), constitutional dyslexia (Skydsgaad, 1942), specific reading disability (Bender and Schilder,

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[1]Arthur L. Drew, "A Neurological Appraisal of Familial Congenital Word-Blindness," Brain, LXXIX, 3 (1956), 440.

1951), primary reading retardation (Rabinovitch, 1955),[2] specific developmental dyslexia (Critchley, 1964, 1970)[3] and others. All of these terms tend to refer to cases of reading disabilities that occur congenitally. At present, European authors frequently use the term "specific dyslexia" and American authors most often use the term "specific reading disability."

There is inconsistency not only in naming this condition, but also in describing it. In the past, only serious cases of non-readers were considered to have specific reading disability. As time went on, the term was also used to describe milder reading problems. This led to added confusion as to the name, the identification, and the origin of this reading disability. In 1968, the Research Groups on Developmental Dyslexia of the World Federation of Neurology, which comprises an international body of experts--neurological, pediatric, psychological, pedogogic--met and drew up two definitions which they recommended for general acceptance. These were as follows:

> Specific Development Dyslexia: A disorder manifested by difficulty in learning to read despite conventional instruction, adequate intelligence and socio-cultural opportunity. It is dependent upon fundamental cognitive disability which is frequently of constitutional origin.
>
> Dyslexia: A disorder in children who despite

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[2]Ibid., p. 449.

[3]Macdonald Critchley, The Dyslexic Child (Springfield, Illinois: Charles C. Thomas, 1970), p. 11.

> conventional classroom experience, fail to attain the language skills of reading, writing and spelling commensurate with their intellectual abilities.[4]

In this paper the operational definition of specific reading disability was used. The children described conform to the following criteria: a failure to learn to read with normal proficiency despite conventional instruction, a culturally adequate home, proper motivation, intact senses, normal or above normal intelligence and freedom from gross neurological defects.[5] A study of the role heredity plays in these reading disability cases follows. This consists of an examination of the descriptive and experimental articles written by European and American authors about causes and symptoms of specific reading disability.

EARLY HISTORY OF "WORD BLINDNESS"

In Europe

In 1896, Morgan, a British physician published a case study describing the reading and writing difficulties of an intelligent fourteen year old boy. This boy confused the sequence of the letters in his own name and made bizarre spelling errors, but did well in mathematics. Morgan noted a similarity between children experiencing a specific reading

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[4] Ibid.

[5] Leon Eisenberg, "The Epidemiology of Reading Retardation and a Program for Prevention Intervention," The Disabled Reader, ed. J. Money (Baltimore: The Johns Hopkins Press, 1966), p. 14.

difficulty and adults that had lost the ability to read because of a brain injury. It was he who first used the term "congenital word blindness" and introduced the ideas of a reading disability as a clinical entity.[6] (The term congenital word blindness is still in common use in Europe today for specific reading disability.)

Although as long ago as 1905, it was observed that word blindness might involve more than one member of a family, this aspect attracted little attention at first. The idea of inheriting a reading problem was investigated by Thomas, who found six patients with this condition within two generations.[7] In the same year, Fisher wrote about congenital word blindness in an uncle and nephew.[8] Stephenson (1907) went so far as to postulate a recessive mode of inheritance on the basis of six cases in three generations.[9] In 1911, Warburg's investigations led him to believe that congenital word blindness was often transmitted by an unaffected mother.[10]

The first extensive monograph on "word blindness"

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[6]Pringle Morgan, "A Case of Congenital Word-Blindness," British Medical Journal, II (November, 1897),1378.

[7]Charles Thomas, "Congenital Word-Blindness and its Treatment," Ophthalmoscope, III (1905), 380-485.

[8]Herbert J. Fisher, "A Case of Congenital Word-Blindness," Transactions of Ophthalmogical Society, United Kingdom, XXX. 1 (1910), 216-225.

[9]Critchley, op. cit., p. 89.

[10]Ibid.

was published in 1917 by Hinshelwood. He devoted one of four chapters to discussing hereditary "word blindness." He reported his observations of cases over a twenty-five year period and concluded that the disability was caused by the malfunctioning of certain brain areas dealing with visual symbols and visual memory and concluded this condition could be inherited.[11]

In the United States

Up to 1920 the descriptive reports on congenital word blindness were found mainly in British Journals. But this condition was also recognized and reported in other European countries and the United States. By 1925 most of the research was being done in the United States.

In 1925 Orton observed "word blindness" in his cases at a mental clinic in Iowa. He wrote that the syndrome was distributed throughout a wide range of intelligence. From close observation of these retarded readers and their efforts at writing and spelling, Orton proposed a theory which centered on the functioning of the brain.[12]

It was Orton who first used the term "specific reading disability." He also coined the term "strepho-symbolia," meaning twisted symbol. This describes what the

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[11]James Hinshelwood, Congenital Word-Blindness (London: H. K. Lewis and Co., Ltd., 1917), pp. 64-74.

[12]Samuel T. Orton, "Specific Reading Disability--Strephosymbolia," Journal of the American Medical Association, XC (April 7, 1928), 1095-99.

child with a reading disability seems to do, namely, twist the order of letters in words and the position of letters on the page. He published the first clear description of the clinical entity for which one uses the term specific reading disability. Orton recognized that certain traits and symptoms occurred with exceptional frequency in the families of patients with specific reading disabilities. These included left-handedness, mixed-handed-eye patterns, speech problems, right-left directional confusion, as well as difficulty with reading, writing and/or spelling by other family members.[13]

In the Salmon Lectures of 1936, Orton summed up his findings which were the basis for his work in the years to come.

> The view presented here that many of the delays and defects in development of the language functions may arise from a deviation in the process of establishing unilateral brain superiority in individual areas, while taking account of the hereditary facts, brings with it the conviction that such disorders should respond to specific training if we become sufficiently keen in our diagnosis and if we prove ourselves clever enough to devise the proper training methods to meet the needs of each particular case.[14]

Since his views were first published, there has been much evidence confirming Orton's work. Herman sees the same clinical syndrome of reading disability with the same

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[13]Samuel T. Orton, Reading, Writing and Speech Problems in Children (New York: W. W. Norton and Co., 1937), pp. 127-30.

[14]Samuel T. Orton, Word-Blindness in School Children and Other Papers on Strephosymbolia, ed. J. L. Orton (Pomfret, Conn.: The Orton Society, Inc., 1966), p. 241.

familial incidence, the same specific nature of the difficulties in performance, the same variation within a normal range, and the same secondary nature of associated emotional problems.[15] DeHirsch and Bender also recognize the Orton syndrome and its inheritance.[16, 17]

SCANDINAVIAN RESEARCH

Scandinavian researchers in particular have published much valuable evidence of the importance of genetics in specific reading disability. Norrie (1939) found genetic tendencies in nearly all his cases. Kagen (1945) stated that he found a genetic relationship in 30 percent of his cases. Ramor (1947) found a familial occurrence of this disorder in 50-60 percent of his cases. Skygaard (1942) published five pedigree charts covering three or four generations which revealed a genetic factor. In his findings he said that no conclusion as to the mode of inheritance could be drawn from such a small family sampling.[18]

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[15]Knud Herman, "Specific Reading Disability," Danish Medical Bulletin, II, 1 (1964), 34.

[16]Katrina DeHirsch, "Specific Dyslexia or Strephosymbolia," Children with Reading Problems, ed. G. Natchez (New York: Harper & Row, 1968), pp. 97-113.

[17]Lauretta Bender, "Problems in Conceptualization and Communication in Children with Developmental Dyslexia," Psychopathology of Communication, ed. P. H. Hock and Lubin (New York: Grune and Stratton, 1958), p. 155.

[18]Bertil Hallgren, "Specific Dyslexia ('Congenital Word-Blindness') A Clinical and Genetic Study," Acta Psychiatrica et Neurologica, Suppl., LXV, 1 (1950), 13-14.

Hallgren's 1950 Genetic Study of Specific Dyslexia

The most important genetic study to date was done in Sweden by Hallgren in 1950 under the title "Specific Dyslexia (Congenital Word Blindness) a Clinical and Genetic Study." This study is widely quoted and footnoted. Hallgren explored the familial incidence and clinical symptoms of 276 children and their families 116 of which were diagnosed as having Dyslexia (specific reading disability).[19] The object of this study was to determine the possible existence of one or more hereditary forms of specific dyslexia, to make a genetic-statistical analysis of the mode of inheritance (if found) and to make a clinical analysis of specific dyslexia with special regard to physical, mental and environmental factors.[20]

Hallgren's sample was made up of children attending the Stockholm Child Guidance Clinic, together with controls from local schools. He divided his sample population into four groups plus a control group: Group 1: Families with the proband (the student affected with specific dyslexia) and both parents affected with specific dyslexia; Group 2: Families with the proband and one affected parent having specific dyslexia; Group 3: Families with both parents unaffected and the cases of specific dyslexia being in the siblings or grandparents; and Group 4: Solitary cases of

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[19] Ibid., pp. 14-16.

[20] Ibid.

the student being the only member of the family affected.[21] For each case a complete family history was taken; tests for vision, hearing, side dominance, intelligence and neurological functions were done. He states that there was no standardized test for dyslexia available but reading and writing tests were given and the severity of the reading retardation and the type of errors made were used as the basis for the child being classed as affected or not. The parents of all of the children used, were given "proof reading tests" and an oral and written spelling test.[22] He wrote up family case histories of the affected children and all borderline cases to explain why they were so classified in his study.

Hallgren found physical illness, neurological disorders, visual and auditory defects in the children with specific dyslexia not to be of significant value when compared with the controls. He concluded that these factors played a small role in the initial cause of dyslexia.[23]

His investigations of the connection between various nervous disorders and specific dyslexia were not conclusive. According to Hallgren the "problem children" are more common among the dyslexic sample than among the control group, but problem children are usually the ones that are brought to

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[21] Ibid., pp. 17-26.

[22] Ibid., pp. 26-38.

[23] Ibid., pp. 47-59.

clinics. Hallgren concluded that it was a failure to perform satisfactorily in a fundamentally important area of communication that caused the emotional problems to develop.[24] He also found no association between specific dyslexia and the incidence of social-economic problems, age of starting school and other environmental factors when compared to the controls.[25] The material was well tabulated and explained in his monograph, but his dyslexic population sample was not a random sample. Therefore, one cannot be sure if environmental factors play an important part in the ability to read, write and spell in some cases of specific dyslexia (specific reading disability).

The relationship between left-handedness and specific reading disability seems to vary from study to study. According to Hallgren, left-handedness occurs more often among dyslexic children than the controls, but no significant difference could be found between the groups. He also found no association between mixed hand and eye dominance and specific dyslexia.[26] American researchers, Spitzer, and Belmont and Birch have also shown that a close relationship between left-handedness or cross dominance (eye-hand-foot) and reading disorders cannot be supported

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[24]Ibid., pp. 92-113.

[25]Ibid., pp. 114-128.

[26]Ibid., pp. 69-82.

with the available evidence.[27, 28] McGlannan, (1968) in her study of "Family Characteristics of Genetic Dyslexia," found left handedness in 70.7 percent of the families and ambidexterity in 58.4 percent of families studied.[29]

Hallgren found a higher incidence of speech defects in children with specific dyslexia when compared to the controls, 10 percent and 1 percent respectively.[30] Herman found speech defects in 23 percent of his dyslexic children and in 2 percent of the controls.[31] This correlates with Hallgren's findings.

Specific reading disability has been observed to affect males more often than females, usually a 3: 1 ratio.[32] Hallgren found rather little inequality in the sex distribution. He felt that because his series were not a random sample of the population this ratio did not hold. His boys out numbered the girls by 76 percent (89 versus 27). Yet there were only 57 percent boys among the poor readers of

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[27]R. L. Spitzer, "The Relationship Between 'Mixed Dominance' and Reading Disabilities," Journal of Pediatrics, LIV, 1 (1959), 76-80.

[28]Lillian Belmont and H. G. Birch, "Lateral Dominance, Lateral Awareness, and Reading Disability," Child Development, XXXVI, 1 (May 1965) 57-71.

[29]Frances K. McGlannan, "Familial Characteristics of Genetic Dyslexia: Preliminary Report From a Pilot Study," Journal of Learning Disabilities, I (March, 1968), 189.

[30]Hallgren, op. cit., pp. 60-68.

[31]Herman, p. 37.

[32]Critchley, p. 71.

the siblings of the affected readers. When he put the parents and siblings together, then 92 percent or 47 percent of the males, and 75 or 37 percent of the females were affected.[33] The difference is still significant. If Hallgren had paid more attention to the uneven sex distribution of his dyslexic students, he may have given more weight to the possibility of sex-influenced manifestation. He dismissed the possibility of sex-linked inheritance because both male and females were affected.[34]

Whether the position of the affected child within the birth order is important was discussed. Hallgren went into the problem of ordinal position much more carefully than previous authors. He found that children with specific dyslexia were evenly distributed among the different numbers in the birth series and thus, birth order could not be a causal factor.[35] Critchley also checked the birth order of 125 random cases of specific reading disability and his findings were the same as Hallgren's.[36]

Hallgren concluded from his study that specific dyslexia (specific reading disability) was determined by an allele (an alternate form of a gene) with an autosomal locus (located on non sex-linked chromosome) with the character

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[33]Hallgren, op. cit., pp. 177-89.

[34]Ibid., pp. 130-31.

[35]Ibid., pp. 190-92.

[36]Critchley, op. cit., pp. 92-93.

being dominant (manifested when one or two allele for the character are present).[37, 38] Hallgren's study is very detailed. He used a large number of subjects who were as randomly selected as possible and who were not affected by other conditions that would introduce confusing results. Every factor which might influence the interpretation of his results was discussed in his paper. Although many consider his investigation novel and disturbing, no one has disproved his main results up to this time.

Hallgren's work and pedigree charts have been used in human genetic books, including Principles of Human Genetics by Stern, to illustrate how "word blindness" can be inherited. But, Stern qualifies Hallgren's findings by saying:

> A trait such as word blindness is frequently not easy to diagnose with certainty--a fact which leaves some doubt regarding its genetic basis. But the existence of specific mental inabilities caused by certain genes is by no means unlikely.[39]

Parks, an Opthalmologist at Northwestern University, raised some questions about heredity as a cause of specific reading disability. He reviewed Hallgren's study and presented a case study to disprove Hallgren's hypothesis. He said that if the hereditary concept is accepted, it would be

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[37] Hallgren, op. cit., p. 213.

[38] Brenda K. Sladen, "Inheritance of Dyslexia," Bulletin of the Orton Society, XX (1970), 30.

[39] Curt Stern, Principles of Human Genetics (San Francisco: W. H. Freeman, 1960), p. 590.

disastrous, for then parents and teachers would consider the condition hopeless and treatment doomed to failure.[40] Many schools and educators have proven him wrong because these children can and are learning today. The case study Parks used had a hereditary eye condition that could be traced for three generations, but the child did not have the characteristics or criteria of a specific reading disability. With glasses and tutoring the child was reading on grade level in one year.[41] Parks misinterpreted Hallgren's monograph by discussing an inherited eye problem and not the inheritance of specific reading disability.

Twin Studies

A different approach to the study of genetic etiology is available through twin studies. It is possible to show that identical twins (identical genetically) have the same abnormalities far more frequently than fraternal twins (genetically similar to non-twin siblings); therefore a genetic influence can be shown to play a more important role in the abnormality than does the environment.[42]

Hallgren's data (containing six pairs of twins--three identical and three fraternal) and Norrie's (1954)

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[40]George E. Parks, "Nurture and/or Nature Cause Reading Difficulties?" Archives of Pediatrics, LXIX (November, 1952), 437.

[41]Ibid., pp. 438-44.

[42]Stern, op. cit., p. 554.

data on twins with "congenital word blindness" were analyzed by Herman. The data indicated that in the three pairs of Hallgren's identical twins, there was concordance (both members of twin pairs had the abnormality) but for only one of the three pairs of fraternal twins were both twins affected. Norrie reported concordance in all nine identical twin pairs, but in only two pairs of the thirty fraternal twins. In both studies there was 100 percent concordance among the identical twins, whereas, the concordance for the fraternal twins was 33 percent. Therefore, he postulated that heredity is a critical etiological factor in specific reading disability.[43]

RESEARCH IN THE UNITED STATES

In the literature on specific reading disability published in this country, Hallgren's monograph is widely quoted. Only a few genetic studies of specific reading disabilities have been published here. Drew has described three cases of familial reading disabilities which exhibit findings similar to those abnormalities present in acquired word blindness due to injury of the parietal lobes of the brain. He suggests that specific reading disability may be due to a disturbance in Gestalt function (a defect in the

[43] Knud Herman, "Congenital Word-Blindness," Acta Psychiatrica et Neurologica, Supplementa CVIII, 1 (1956), 180-84.

visual motor field) and inherited as a dominant trait.[44]

DeHirsch and Bender noted the varying occurrences in reading disabilities of defects in directional selection, auditory-visual-phonetic disintegration, spatial disorientation and reversals. They interpreted these defects as Gestalt disturbances, and therefore, thought of specific reading disability as failure in Gestalt functions.[45, 46] Rabinovitch expressed a similar concept of specific conceptual deficiency, disorientation and body image. He calls these "core layers" underlying reading disability.[47]

Drew theorized that a defect in Gestalt recognition may be due to failure, or delay in maturation of some functions of the parietal lobes. The anatomical physiological substrate might still be a failure to develop complete dominance because of inherited factors, whose manifestations are not consistently manifested. This could account for the many patterns of language dysfunction, the nature and intensity of the characteristics in each individual that depends on other constitutional and environmental influences. Drew also felt that a definite cerebral defect caused by a brain injury or malfunction can not be made to

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[44] Drew, pp. 456-58.

[45] DeHirsch, pp. 231-45.

[46] Bender, p. 155.

[47] Ralph D. Rabinovitch, "Neuropsychiatric Considerations in Reading Retardation," Reading Teacher, XV (May 1962), 433-38.

fit the facts of familial incidence and the failure to demonstrate any other consistently associated neurological impairment.[48]

To explain his theory, Drew described three family units in which he reported family history and results of reading and psychological tests administered to each family member. He reported that his results support the hypothesis that specific reading disability was inherited in these three families.[49] Whether or not "Gestalt functions" prove to be the underlying cause of the maturation failure must still be explained and be proven by future research.

In 1965 Walker and Cole undertook a statistical study of specific reading disabilities. The sample selected consisted of families with three children, all presently in a suburban Boston Public School. If the I. Q. was below ninety in any one of the three siblings in a family, the family was not used. Seventy-five family units remained. The suburban school system and method of collecting data were discussed. Specific reading disability was defined, for the purpose of this study, as spelling performance below normal standards in students with normal intelligence and selection by the school at an earlier date for individual remedial help because of poor reading performance.

The Stanford Spelling Test was used as the diag-

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[48]Drew, loc. cit.

[49]Ibid., pp. 441-48.

nostic instrument to determine spelling proficiency. Four reasons were given for using the spelling rating alone. Among them was that poor spelling is the single most universally present defect in specific reading disability. According to their definition 25.3 percent of the children tested showed specific reading disability. The incidence of the disorder was shown to be much higher in certain families than in others, with clear sibling aggregation. Using the Chi square to test the hypothesis, they found the prevalence of this disability in certain families to be significant at the .005 level.[50]

The authors acknowledge that before coming to any conclusions from the study, a larger and more varied group of children must be studied. If the authors' data are valid, then the factor of familial co-incidence may be interpreted to mean that the disability was inherited. Their definition of specific reading disability and way of determining the affected students does not agree with criteria set forth in other studies. Therefore, this high percentage of affected students should be questioned. But the data presented seem incompatable with the hypothesis that specific reading disability is caused by cultural deprivation or economic status, psychogenic factors, method of teaching, low intelligence,

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[50]Louise Walker and Edwin Cole, "Familial Patterns of Expression of Specific Reading Disability: Part 1, Prevalence, Distribution and Persistence," Bulletin of The Orton Society, XV (1965), 12-17.

brain damage, childhood diseases and delayed maturation in the sense of something which is out grown.[51] The conclusion that environmental factors do not cause specific reading disability but can aggregate it is in agreement with earlier authors already cited.

Silver studied fifty-six children with specific reading disability. He collected data on prenatal and perinatal difficulties, medical problems, and family history of similar learning difficulties (for mother, father, and siblings). In the total study, 39.4 percent of the children had a positive family history of similar learning difficulties. In each of the families with a positive history of reading disabilities the history suggestive of central nervous system stress was made less significant by the total family data. This stress was not a contributing factor in producing this syndrome. Even though there was a history of prenatal, perinatal or postnatal difficulties, siblings without a history of such difficulties also had reading problems. This study strongly suggests that the etiologic factor with some of the children with specific reading disability is an inherited central nervous system dysfunction rather than that of brain damage.[52]

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[51] Ibid., pp. 16-23.

[52] Larry B. Silvers, "Familial Patterns in Children with Neurologically-Based Learning Disabilities," (paper to be published in Journal of Learning Disabilities in 1971).

CHARACTERISTICS OF SPECIFIC READING DISABILITY THAT MAY BE INHERITED

The manifestations of specific reading disability are seen in the difficulties the child has with the written word. The search of the literature revealed that the investigators found the following characteristic symptoms in cases of specific reading disability in their studies. The fact that many of these same characteristics are found in very young children and beginning readers is normal, but in young and beginning readers they disappear by the end of the primary grades. However, for the child with specific reading disability, the characteristics are more pronounced and remain with him for a longer period of time. Some of these difficulties remain with him into adult life.[53] But not all children have exactly the same characteristics. Rabinovitch summarizes the situation when he ascribes to the child with specific reading disability a "characteristic pattern, with much variability from patient to patient."[54]

The intelligence and achievement functioning of these children as described in the literature are characterized by relatively higher ratings in performance skills than in verbal skills on the WISC. The occurrence of significantly poorer verbal than performance skills is an

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[53]Knud Herman, "Specific Reading Disability," pp. 34-35.

[54]Ralph D. Rabinovitch, "Reading and Learning Disabilities," American Handbook of Psychiatry (New York: Basic Books, 1959), pp. 867-69.

expected finding in a representative sample of children with specific reading disability. Low scores in information, arithmetic, digit span and coding patterns appear to characterize this group of poor readers.[55, 56, 57] Not every child conforms to this pattern. Burt (1966) designed models of modes of inheritance to describe the population distribution of intelligence as measured by intelligence tests. He did not specifically consider reading problems, but he concluded that: (1) there is ample evidence of nonrandom mating in regard to intelligence; (2) multifactorial and unifactorial components are largely concerned with rare defects; and (3) there are slight indications of sex--linkage.[58] Burt's theories should be investigated further and extended to include specific reading disabilities.

Familial Similarities of the Characteristic Symptoms

At Princeton University, an investigation of similarities in parent-child test scores for evidence of hereditary components was undertaken. This study examined 104

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[55]Pauline Adams, "Patterns of Intellectual Functioning in Learning Disability Children and their Siblings Compared with Successful Students and their Siblings," Bulletin of the Orton Society, XVIII (1968), 40-48.

[56]McGlannan, pp. 186-88.

[57]Cyril Burt, "The Inheritance of Mental Ability," American Psychologist, XIII, 1 (1958), 1-15.

[58]Ibid.

family units using eight psychological tests. In fifty--eight tests, word association was the only variable which did not show significant similarity between parent and child. Spatial visualization and reasoning ability showed a unique family correlation pattern. Perceptual speed and music aptitude clearly fulfilled the requirements that best fit the autosomal genetic model with approximately the same percentage for the father-son distribution of scores as with the mother-daughter scores.[59]

Perceptual-motor functioning difficulties in varying degrees are noted in children with specific reading disabilities. Auditory imperception (a disorder in which a defect in the recognition of sound occurs without hearing loss) and defective visual memory of word parts, (a disorder in which the child has normal eye sight but difficulty remembering letter and word forms) have been noted as characteristics in these children.[60, 61] So far, no conceptual model has been successful in describing exactly what goes on between the stimulus and a response within the child. Until the process has been precisely described, and tests designed

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[59]Richard Stafford, "An Investigation of Similarities in Parent-Child Test Scores for Evidence of Hereditary Components," Eric/Crier, IV (July 1969), 75.

[60]Herbert G. Birch and L. Belmont, "Auditory Visual Integration in Normal and Retarded Readers," American Journal of Orthopsychiatry, XXXIV (October 1964), 852-61.

[61]Marianne Frostig, The Frostig Program for the Development of Visual Perception (Chicago: Follett Publishing Co., 1964).

accordingly, no one knows where or why there is a malfunction.

Birch and Belmont studied the relationship of auditory-visual integration to reading retardation in 200 nine and ten year old children. One hundred and fifty were retarded readers and fifty were normal readers. The retarded readers were significantly less able to make judgments of auditory-visual equivalence than the normal readers. When children with low normal I. Q's were eliminated from consideration the significant difference in auditory-visual test performance between the retarded and normal readers was sustained. They interpreted the findings as indicating that auditory-visual integration has specific relevance to reading, although it is not the sole factor underlying reading incompetence.[62]

Forrest studied the relationship of neurological and medical factors in children with specific reading disability and their families. The three groups of subjects that were matched by I. Q., sex and age consisted of seventy-six children with specific reading disabilities, seventy-six of their siblings and seventy-six controls. He tested their ability to reproduce auditory tapping patterns and found a significant discrepancy between the controls and the children with reading difficulties. However, the ability of children

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[62]Birch, loc. cit.

with reading problems and their siblings was remarkably similar. He noted that this task showed a close correlation to the digit span score of the WISC which showed similar diagnostic and familial importance.[63] He also found that right and left discrimination, fast alternating hand movements, poor listening skills after age two, and ease of mother-child communication were significant differentiating factors at the .01 level. Double simultaneous touch (face--hand test) and fast alternating finger movements showed a significant differentiation at the .05 level. He found that measure of hand, foot, eye preference, arm extension test, walking on a balance beam were not significant factors between the groups.[64]

Directional Orientation

The children with specific reading disability have difficulty differentiating letters. This may be regarded as problems with positioning of certain elements in space, the arrangement of letters in a word and a phrasal sequence difficulty. Because of this problem, many researchers are studying the disintegration of spatial functions and the disorganization of temporal functions. A larger number of these efforts deal with performance on right-left orien-

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[63]Thomas Forrest, "Neurological and Medical Factors Discriminating between Normal Children and Those with Learning Disability," Bulletin of the Orton Society, XVIII (1968), 48-54.

[64]Ibid. pp. 49-50.

tation tasks.

Investigation of this factor has proceeded along two lines. First, the ability to discriminate between different orientations of identical figures and the tendency to reverse the conventional left to right orientation as well as temporal orientation in sequential stimulation have been assessed. Secondly, since directional sense has been related to the development of the body schema, the right--left orientation of normal children and children with specific reading disability has been examined.[65] Benton in his controlled studies found that a significantly higher percentage of the children with reading problems have a right-left orientation problem than the controls.[66] Silver and Hagin found similar results.[67] Disordered directional sense is considered by Critchley to be a true disturbance in spatial orientation and not a verbal or semantic defect.[68]

Herman and Norrie in 1958 put forward the hypothesis that specific reading disability was based on the same fundamental disturbance responsible for the Gertsmann's syn-

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[65]Arthur L. Benton, "Dyslexia in Relationship to Form Perception and Directional Sense," Reading Disability: Progress and Research Needs in Dyslexia, ed. J. Money (Baltimore: The Johns Hopkins Press, 1967), p. 96.

[66]Ibid., p. 99.

[67]Archie A. Silver and Rosa Hagin, "Specific Reading Disability, Delineation of the Syndrome and Relationship to Cerebral Dominance," Comprehensive Psychiatry, I (April 1960), 126-34.

[68]Critchley, p. 50-64.

drome (an inherited condition causing disturbances in directional function). However, since not all the conditions for this syndrome are found in children with specific reading disability, their hypothesis had to be rejected.[69]

Cerebral Dominance

Children with specific reading disability who were not strongly right handed were recognized early in the history of "congenital word blindness." Increasing importance is being attached to this aspect of the problem as more investigators note the prevalence of left handedness and mixed dominance among affected children.[70] Gooddy and Reinhold attributed these reading disabilities to a cerebral defect that may be related to a too close similarity of function, that is a lack of asymmetrical function of the two hemispheres of the brain.[71]

The greater frequency of cerebral ambilaterality in children with specific reading disability has led Zangwill to suggest, "the genetic factors which predispose one to dyslexia relate not to a specific psychological capacity but

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[69]Knud Herman and Edith Norrie, "Is Congenital Word--Blindness a Hereditary Type of Gerstmann's Syndrome?" Psychiatrie und Neurologie, CXXXVI (1958), 59-73.

[70]Critchley, op. cit., p. 65.

[71]William Gooddy and Margarett Reinhold, "Congenital Dyslexia and Asymmetry of Cerebral Function," Brain, LXXXXIV, 1 (1961), 240-42.

to the general determination of handedness and cerebral dominance."[72]

The idea of cerebral dominance has two aspects which have not always been clarified in the literature. In the first place, the question of handedness is a much more complex problem than is generally understood. Even after a battery of tests to determine handedness, the results may often be relative. The second point is the fact that in correlating handedness with reading disability, it has often been only too obvious that the author was using a group made up of diverse types of poor reading ability, not all meeting the criteria for true cases of specific reading disability.

Zangwill had pointed out that only some il-lateralized children have reading problems, therefore, he suggested three possible explanations: (1) poorly developed laterality and reading defects could both be due to the effect of an acute cerebral lesion; (2) reading difficulty and the lack of cerebral asymmetry could both be taken as evidence of an inherited maturational lag; and (3) children who lack firm lateral preferences are particularly vulnerable to the effects of stress.[73] Research that utilizes

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[72] O. L. Zangwill, Cerebral Dominace and Its Relation to Psychological Function (Edinburgh: Oliver and Boyd, 1960), p. 116.

[73] O. L. Zangwill, "Dyslexia in Relation to Cerebral Dominance," Reading Disability: Progress and Research Needs in Dyslexia, ed. J. Money (Baltimore: The Johns Hopkins Press, 1967), p. 105.

data from well controlled experiments is needed before any conclusions can be drawn about the relationship of cerebral dominance and specific reading disability.

BIOCHEMICAL DISORDERS THAT MAY ACT AS CONTRIBUTING FACTORS IN READING DISORDERS

No single biological system operates independently. The nervous system and the endocrine system compliment each other. The nervous system controls and regulates the activity of the endocrine system and the endocrine system in turn alters the activity of the nervous system. Since the act of reading involves the nervous system, the endocrine system facilitates or hinders the reading process.

Smith and Carrigan Study

Smith and Carrigan theorized that severely retarded readers that are characterized by blending deficiency, abnormally low reading rate on familiar reading material, deficient discrimination of sounds and visual symbols have a neurochemical imbalance causing a synaptic transmission impairment.[74] To make normal neural transmission possible it is essential that there be a balance between two neurochemicals, acetylcholine (ACH) and cholinesterase (CHE). ACH is needed if the impulse is to bridge the junction (synapse) between certain neurons. CHE acts as a circuit

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[74]Donald E. Smith and Patricia M. Carrigan, The Nature of Reading Disability (New York: Harcourt, Brace and Co., 1959), p. 14.

breaker and neutralizes the ACH and stops the transmission from firing.

Smith and Carrigan contended that an overabundance of ACH made it difficult for an individual to change his fixation point, resulting in slow reading, an inability to blend phonemes, and inadequate auditory and visual discrimination. Oh the other hand, too much CHE makes it difficult for an individual to sustain adequate fixation. When this condition exists, the individual would tend to be a fast inaccurate reader.[75] Endocrine anomalies may influence synaptic transmission. The thyroid gland especially affects the endocrine system. For example, it has been observed that some types of abnormal mental development are know to be functionally related to thyroid disease (e.g., cretenism). One of the thyroid hormones is a determinant of cell metabolism.[76]

Smith and Carrigan's study consisted of three stages: the first was the diagnosis of disabled readers by means of reading and psychological tests in a public school system; second, a clinical study of suspected endocrine problems from the same population was undertaken; third, the subjects were divided into groups, some groups were treated with one or more of the following: vitamins, hormones, (thyroid) stimulants and/or tranquilizers plus control groups receiving

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[75]Ibid., pp. 15-20.

[76]Ibid., p. 23.

placebo pills and an untreated group. A complete description of the theory, testing, experimentation and results were given. The results indicated no significant difference among the groups in reading gain or in psychological test scores. Their evaluation of this experiment points to the need for better methods of testing and measuring this type of data. They felt their study was out of date before it was even completed and a new one should be undertaken.[77] This study was published in 1959.

Other Biochemical Studies

Eames (1959) did a study comparing twenty-four reading failures with endocrine dysfunction, one hundred reading failures without endocrine disturbances and one hundred controls. The paper described the subjects and the effect that thyroid deficienty (hypo-thyroidism), excess thyroid (hyper-thyroidism) and pituitary deficiency had on general health and on school work and reading. He found the most common endocrine disorder in poor readers to be hypo--thyroidism.[78]

McGlannan (1966) undertook a study to identify and delineate some of the genetic characteristics of families in which there was a child with specific reading disability. The sample population of the study was composed of three

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[77]Ibid., p. 23-91.

[78]Thomas H. Eames, "The Effect of Endocrine Disorders on Reading," Reading Teacher, XII (April 1959), 263-65.

generations of sixty-five families. The criteria used for sample selection, the test instruments, the administration and observations were closely controlled and are clearly defined in her paper. She reported 75.6 percent of the families had diabetes or low blood sugar, 76.9 percent had allergies, 70.7 percent had left handed people, 58.4 percent reported ambidexterity. She did not report these factors as they relate to each family unit. She sets forth the hypothesis that there exists a "vulnerable family" syndrome and it is these families with specific genetic characteristics, which are most likely to produce a child who suffers from specific reading disability. A more extensive study was undertaken to prove this hypothesis but the results are not yet available.[79]

The role that the bio-chemical factors play in specific reading disability has yet to be proven or disproven. There is evidence that learning disabilities are related to steroid insufficiency, and also to impaired protein synthesis at the level of DNA transmission.[80]

The new technique of counting and visualizing chromosomes within the cells has revealed no suggestions, so far, of a connection between chromosomal aberration and reading disability. But little research on chromosomes and

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[79] McGlannan, pp. 185-90.

[80] Albert O. Rossi, "Genetics of Higher Level Disorders," Journal of Learning Disabilities, III, 1 (August 1970), p. 387.

genes as they relate to specific reading disability has been done. However, some chromosomal disorders do have a definite affinity with mental deficiency.[81, 82]

SUMMARY

There are many family case studies reported in the literature that show several members of a family affected by specific reading disability. Critchley has written a comprehensive description of specific learning disabilities with emphasis on the etiological relation to heredity, cerebral immaturity and confused dominance.[83] There are other studies but the most widely quoted and comprehensive studies have been discussed in this chapter. The characteristic symptoms of specific reading disability that have a hereditary tendency were described. Some of these studies are non-conclusive and much more investigation is needed.

As yet, no known study has been done to obtain familial information about specific reading disability using a questionnaire containing genetic characteristics of the disability. There are still unanswered questions about the

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[81] M. A. Ferguson-Smith, "Chromosomes and Human Disease," Progress in Medical Genetics (New York: Grune and Stratton, 1961), pp. 292-334.

[82] A. R. Sohval, "Recent Progress in Human Chromosome Analysis and Its Relation to the Sex Chromatin," American Journal of Medicine, XXXI, (1961), 397-441.

[83] Macdonald Critchley, The Dyslexic Child (Springfield, Illinois: Charles C. Thomas, 1970), p. 11.

relationship between heredity and specific reading disability. Hallgren's single gene theory should be investigated further. Also the bio-chemical factors that are inherited should be examined to see if they act as a contributing factor to the reading problem.

Little has been done since the Hallgren study of 1950 to test the genetic origin of specific reading disability. Conclusive evidence on the familial aspects of this disorder is needed. Therefore, this study was undertaken as another step in the collection of such evidence.

Chapter 3

DESIGN AND METHOD OF STUDY

The purpose of this study was to investigate the relationship between heredity and specific reading disability. The first task was to design a questionnaire to obtain information on family history. This questionnaire, called the Family Check List, was designed to be used as an added tool in getting further information about the child with a reading problem. The Family Check List was tested on a group of students diagnosed as having specific reading disability, and a control group of average and better readers. From this information, a clinical and genetic--statistical analysis of specific reading disability cases was performed.

DESCRIPTION OF THE STUDENTS USED IN STUDY

All the students in this study had an I. Q. of one--hundred or above, came from culturally adequate homes, were attending public school regularly, had good vision and hearing, and showed no signs of gross neurological defects. The students were selected from three populations. The children of one clinic group had been diagnosed by the University of Pennsylvania Reading Clinic. These children were at

least ten years old and were reading on a primer level. The size of this group was four. This group will be designated hereafter as P.C. for the University of Pennsylvania Reading Clinic group. Another clinic group was made up of children who had come to the Glassboro State College Reading Clinic because of reading problems. The researcher selected those cases from the files which fit the description of specific reading disability. These children had reached the average age of ten years and were reading at least two years below their expected reading level. Their records noted a perceptual difficulty, letter reversal problem, a difficulty hearing vowel sounds, and/or a limited sight vocabulary. The size of this group was twenty-four. The group will be designated hereafter as G.C. for the Glassboro Reading Clinic group. The third group was a control group selected from a third, a fourth, and a fifth grade class at Bell Elementary School in Blackwood, New Jersey. The classes were homogeneously grouped by I. Q. and achievement. The classes having average or above average I. Q. were used for this study. Every third child on the alphabetized register was selected. The register was split by sex and equal selections of boys and girls were made. The size of the control group was twenty-five.

THE INSTRUMENT USED: THE FAMILY CHECK LIST

Rationale of Design

Most of the information gathered for the familial

histories discussed in the review of the literature, was obtained from retrospective personal interviews. The reliability of this type of information is questioned today. Interview responses to questions about the incidences of family learning problems may be especially vulnerable to distortion.[1]

The researcher was unable to locate a questionnaire, check list, schedule, or form that could be used to obtain family background information relevant to genetic characteristics and reading disabilities. Questionnaires are available to obtain information about a child's pre-natal, birth, and early years of development. Neurological, psychological, and medical forms are also available. Therefore, it was necessary, as part of the design of this study, to devise a questionnaire that contained specific questions relating to family members. It was mailed to the home and filled out at the convenience of the family members.

From the search of the literature, a pattern of symptoms related to specific reading disabilities was found. Questions were devised to elicit the needed family genetic background related to these symptoms. The fact that many of these symptoms are found in many young children and beginning readers is normal. These problems for the most part disappear as the child ends the primary grades. But in the

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[1]L. C. Robbens, "The Accuracy of Parental Recall of Aspects of Child Development and of Child Rearing Practice," Journal of Abnormal Social Psychology, LXVI, 1 (1963), 261-70.

child with specific reading disability, the reading problem emerges more clearly as the child progresses in school and is required to accomplish more demanding tasks. A significant number of these difficulties remain with him into adult life.[2]

The Construction of the Family Check List

The Family Check List was constructed following an examination of the research literature. Those characteristics most often linked to specific reading disabilities were summarized and formed the basis of the thirty-four items which made up the questionnaire used in this study. The Family Check List is shown in Appendix A. Different types of questions were asked: (1) to get an over-view of the reading problem; (2) to check the characteristics of specific reading disability that may be present in a family unit; and (3) to check the reliability of answers given on the check list. Only those questions were asked which could be understood by the average adult.

The relationships of family members is needed for any genetic study. Therefore, each family member was listed in the check list including adopted and twin members, although family members who were adopted and not related by blood lines could not be used. Identical twins were

[2]Knud Herman, "Specific Reading Disability, with Special Reference to Complicated Word Blindness," Danish Medical Bulletin, XI (May 1964), 34-35.

expected to show the same patterns if the check list was completed correctly.

The questionnaire was made up of five groups of questions plus a Draw-A-Man Test. One group of questions dealt directly with information concerning the problem with written words (questions nine through fourteen). These questions concerned difficulty with reading, spelling, writing, and foreign languages. The second group of questions pertained to difficulties encountered in reading (questions fifteen through twenty-three). These included difficulties with visual discrimination, auditory discrimination, reversal in sequence of letters and numbers, visual memory, auditory memory, spatial visualization, perceptual speed, and other associated learning problems. Some persons with reading difficulties experience directional confusion, therefore question twenty-four was included. The topic of mixed cerebral dominance was noted in the literature as a possible cause of reading problems. As stated in Chapter 2, research in this area has not been conclusive. Therefore, questions thirty-one and thirty-three were included to learn if mixed dominance or left-handedness played a role in the reading difficulties studied here. The fifth group of questions was devised to reveal information about symptoms of endocrine disorders in the population studied. Endocrine disorders may act as a contributing factor in reading disorders and may account for the change from day-to-day in the severity of the symptoms observed in a child with specific

reading disability. Questions twenty-five through thirty are related to these factors. These questions elicited information about the families' thyroid conditions, diabetes, low blood sugar, and allergies. All these questions were written so that the average adult would be able to understand and answer them. A place was left next to each question for additional comments.

The final part of the questionnaire asked the student to Draw a Man and write a sentence about the man. The Draw-A-Man Test provides standards which can be used to measure the intellectual maturity (correlated to the WISC performance scores) of the child.[3] The purpose of the sentence was to give some indication of his other language abilities.

DATA COLLECTION

The children with specific reading disability in the U.P.C. and G.C. groups had already been diagnosed by the University of Pennsylvania and Glassboro Reading Clinics respectively. Information pertaining to home, school, and medical background was obtained by these institutions. Results of reading tests, WISC scores, and psychological tests had already been gathered and were available in each child's folder. The Family Check List was used to gain

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[3]Pauline Adams, "Patterns in Intelligence Functions in Learning Disability Children and Their Siblings," Bulletin of Orton Society, XVIII (1968), 40-48.

family history which was related to reading disorders. It was devised and used as an added tool in diagnosing reading problems.

The Check List was mailed to the parents of the children in all three groups. After completion, the parents were requested to mail them back to the investigator. To increase the reliability of the answers, no last names or addresses were put on the forms. Each case was identified by a code number.

The main focus of this study was on relationships between hereditary factors and specific reading disability. Therefore, all children who were adopted were excluded from the study because of the impossibility of obtaining the necessary history.

EVALUATION OF THE DATA

The information received on each individual was analyzed to see if enough characteristic symptoms were present to determine if that person could be classified as affected by specific reading disability. First, a difficulty with the written word must be noted on the Check List. Then the following characteristics of specific reading disability, as revealed by the literature were checked: difficulty with visual discrimination, visual memory, auditory discrimination visual memory, auditory discrimination, auditory memory, reversals in sequence of letters and numbers, spatial visualation, perceptual speed, and/or left-right confusion

confusion (questions fourteen through twenty four). The possibility of setting forth one set of symptoms for identifying specific reading disability, is almost impossible because specific symptoms very from person to person. Rather, when the symptoms which characterize a person's reading disability are taken as a group, a pattern of symptoms can be seen.

The remaining questions were informational only. They were included to give a more complete picture of the familial background that may affect this condition.

STATISTICAL ANALYSIS OF DATA

For the statistical analysis the families were grouped as follows:

Group 2-A Families where both parents experienced a specific reading disability. (two affected parents)

Group 1-A Families where only one parent experienced a specific reading disability. (one affected parent)

Group 0-A Families where neither parent experienced a specific reading disability, but cases were noted in siblings or grandparents. (zero affected parents)

Group C-A Just the child being studied experienced specific reading disability. (Child only)

The data were then analyzed to test the null hypo-

thesis.

For the Mendelian Genetic analysis, the Weinberg proband method[4, 5] was used to statistically adjust the data. All families with only one proband (affected child) over the age of seven were omitted from this analysis. This helps to correct for sample bias. This method assumes that the affected parent is a hybrid (heterozygous) and therefore carries the gene for the disorder. Chi square tests were performed to determine if the resulting percentages from the Weinberg method were statistically significantly from the theoretical fifty percent in a single hybrid autosomal dominant mode of inheritance. The data were analyzed to see if the sex of the affected person, position in the birth series and/or biochemical factors play a role in reading disabilities.

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[4]Bertil Hallgren, "Specific Dyslexia ('Congenital Word Blindness') A Clinical and Genetic Study," Acta Psychiatrica et Neurological, Supple., 65, 1 (1950), 200.

[5]Curt Stern, Principles of Human Genetics, (San Francisco: W. H. Freeman, 1960), p. 143-73.

Chapter 4

ANALYSIS OF DATA

It was the purpose of this study to investigate the relationship of heredity to specific reading disability. To obtain the needed family background information for this study a questionnaire, called the Family Check List (see Appendix A), was mailed to sixty-three families. The parents were asked to fill it out and return it to the investigator. From this information a clinical and genetic--statistical analysis was made.

FAMILY CHECK LIST RETURNED

Sixty-three check lists were mailed to families; fifty-three, or 84% were returned. Table 1 presents the number of check lists returned by the population sample. Of the total number mailed, twenty-nine were control families with a return of twenty-five; twenty-six were sent to Glassboro Clinic families with a return of twenty-four; and eight were sent to University of Pennsylvania Reading Clinic selected families with a return of four. One of the University of Pennsylvania families had to be eliminated from the study because the child in the study was adopted.

All data from each check list were then tabulated on

the basis of each individual and of each family. The Glassboro Clinic Group and the University of Pennsylvania Clinic Group were combined for some of the analyses, giving twenty--seven families that had at least one child diagnosed as having specific reading disability. There were twenty-five families in the control group.

Table 1

Family Check Lists That Were Mailed, Returned and Used in This Study

Group	Total Mailed	Total Returned	Returns Used in Study
Controls	29	25	25
Glassboro Clinic	26	24	24
University of Pennsylvania Clinic	8	4	3
Totals	63	53	52

Table 2 summarizes the number of persons used in this study. Information was also gathered about the child's grandparents but this was not used in the data analysis. There were fifty-two students used in this study. Their name appeared on the top of the Family Check List when it was mailed home. These students nad 101 brothers and sisters over the age of seven years and 104 parents. Thus, the total number of persons used in this study was 257.

Table 2

Number of Persons Used in the Data Analysis

Group	Students	Siblings	Parents	Total
Controls	25	39	50	114
Glassboro Clinic	24	46	48	118
University of Pennsylvania Clinic	3	16	6	25
Totals	52	101	104	257

SUMMARY OF CHARACTERISTICS OF SPECIFIC READING DISABILITIES, AS SHOWN IN THE FAMILY CHECK LIST, AS REPORTED BY THE CONTROL AND CLINIC GROUPS

Table 3 summarizes items of the Family Check List as they were reported by families and by students in this study. The first four columns compare the characteristics that were present in the families of the control group (twenty-five) and of the families of the clinic group (twenty-seven), and in the family members other than the student studied. The last two columns compare the characteristics that were present in just the students of the control (twenty-five) and clinic (twenty-seven) groups. Items nine throuth thirteen give an over-view of the difficulties encountered with the written word; items fourteen through

twenty-four summarize the characteristics of specific reading disability as shown on the Check List.

All characteristics of specific reading disability used in this study were present in both the control and clinic families. But items nine through twenty-three were present more often and in greater number in the clinic group than the control group.

When the clinic and control groups of students were compared there was a difference in the number of times each characteristic was reported for the two groups. Reading and spelling difficulties were reported in almost all cases of the clinic groups compared to three or less in the control group. A larger speaking than reading vocabulary, confusing sounds of letters, difficulty with alphabetic order, reading very slowly and omitting words were reported in fifteen or more of the clinic students compared to one or no indication in the control students. Poor handwriting, speech problems, reversal of letters and concentration on groups of numbers were reported at least eight times in the clinic cases, compared to one or a no response in the control cases. The response to difficulty with a foreign language and right and left confusion showed no significant difference in the number of times reported, both being less than two in the clinic and control groups.

Table 3

Summary of Characteristics of Specific Reading Disability, as shown in the Family Check List,* Comparing the Control and Clinic Families; the Control and Clinic Students

Characteristic		Present in Family (25) Control +stu	Present in Family (25) Control -stu	Present in Family (27) Clinic +stu	Present in Family (27) Clinic -stu	Present in Student (25) Control	Present in Student (27) Clinic
9/10	Reading Difficulty	6	6	27	22	3	27
11	Spelling	10	10	26	21	1	26
12	Poor Hand-writing	10	9	16	14	2	9
13	Foreign Language Difficulty	4	4	8	8	0	1
14	Larger Vocabulary	5	4	14	9	1	15
15	Speech	8	5	11	7	1	8
16/18	Reversals	3	2	19	7	0	8
17	Letter Sounds	2	2	19	7	0	17
19	Codes	2	2	7	5	0	4
20	Digit Span	6	2	12	10	1	7
21	Alphabet	1	1	14	7	1	12
22	Reads Slow	8	8	18	11	1	15
23	Omits Words	7	6	17	8	1	17
24	Left-Right Confusion	3	3	3	2	0	2

*See Appendix A

GENETIC STATISTICAL ANALYSIS

The families used in this study were divided into four main groups on the basis of the occurrence of reading disabilities in the family. Affected (A) refers to that person having a reading disorder that met the criteria for this study, (M) for male, (F) for female. A Proband means the affected student used in this study. A summary of each family used in this study is given in Appendix B. The main groups are:

Group 2-A Families where both parents of the child studied were affected with reading disability.

Group 1-A Families where only one parent of the child studied was affected with a reading disability.

Group 0-A Families where neither parent of the the child studied was affected with a reading disability, but where cases were noted in siblings of the child.

Group C-A Just the child being studied is affected with specific reading disability.

Distribution According to Sex

Table 4 shows the sex distribution of the cases of specific reading disability in the children studied in both clinic groups. The total number of affected males is greater than that of the famales. The analysis of the data shows that twenty-three males and four females, or a ratio of five boys to one girl, were present in the ccmbined clinic population. In the Glassboro Clinic sample there

were twenty males to four females or a ratio of five to one. When the probands of both clinics were combined with their affected siblings the total number of forty-one males and six females or a ratio of six to one was again noted. When the probands of the Glassboro Clinic and their affected siblings were tallied, thirty-three males to six females were noted, or a ratio of five to one. The above figures show a greater proportion of males than females, in this study had specific reading disability.

Table 4

Distribution According to Sex of the Clinic Students and their Affected Siblings

Group	Proband Male GC	Proband Male PC	Proband Female GC	Proband Female PC	Siblings Male GC	Siblings Male PC	Siblings Female GC	Siblings Female PC	Total Male GC	Total Male PC	Total Female GC	Total Female PC
2-A	1				1				2			
1-A	10	3	3		9	5	1		27		4	
0-A	3		1		3		1		6		2	
C-A	6								6			
Total	23	3	4	0	13	5	2	0	41		6	
	M-23		F-4		M-18		F-2		M-41		F-6	

Table 5 shows the distribution of affected students, affected siblings and affected parents in the combined clinic groups. When the sex of the affected parent was tallied with the affected student and siblings, fifty-two

males to thirteen females, or about four males to one female. When only group 1-A (one affected parent) is considered the ratio is about four to one.

Table 5

Distribution of Both Clinic Students, their Affected Siblings and Affected Parents According to Sex

Group	Proband M	Proband F	Sibling M	Sibling F	Parents M	Parents F	Total	Ratio M	Ratio F
2-A	1	0	1	0	1	1	4	3	1
1-A	13	3	14	1	10	6	47	37	10
0-A	3	1	3	1	0	0	8	6	2
C-A	6	0	0	0	0	0	6	6	0
Total	23	4	18	2	11	7	65	52	13
Ratio								4:1	

When all the children in this study, regardless of age and whether affected with a reading disability or not, were categorized by sex, the following ratios were obtained. In the control group there were forty-one males and forty females or a ratio of approximately one to one, as found in the normal population. In the clinic (1-A) group there were forty-four males and twenty-two females, or a ratio of two males to one female. Fifty percent of these males were affected and 20 percent of these females were affected. If you remove the students used in this study, from this tabulation, you find the ratio of one to one remaining in

the controls. There is still a greater number of boys than girls in the clinic group. In group A-1, the ratio is now thirty-one boys to nineteen girls. This fact should be investigated further.

The Position of the Affected Child in the Birth Series

In the twenty-seven clinic families used in this study there were eighty-nine children. Children that had not reached the age of manifestation (greater than seven years old) were not used in the tabulation. Families having only one child, where this only child was affected, were included in the following calculations.

Table 6 shows the distribution of the children with specific reading disability according to their position in the birth series. The affected children are distributed among the different positions in the birth series as follows: twelve were first born, twelve were second born, four were fourth and six were fifth born. Birth order does not appear to be a contributing factor in the cases studied.

Mendelian Analysis

Fifty-two families were analyzed for the role of heredity in specific reading disability. In only one family were both parents affected by a reading disability (group 2-A). Both of their children that had reached the age of manifestation had been diagnosed as having specific reading disability. There was one son, age six, that could not be

Table 6

Position in the Birth Series of Children with Specific Reading Disability in the Combined Clinic Groups

No. of Children Age 8 or Older	No. of Families	Affected Children	No. Affected Children in Order							
			1	2	3	4	5	6	7	8
1	1	1	1							
2	10	16	9	7						
3	7	12	1	4	7					
4	1	2			1	1				
5	6	12	1	1	4	2	4			
6	1	1					1			
7	0	0								
8	1	3				1	1			1
Total	27	47	12	12	12	4	6	0	0	1

included in this study because he had not reached the age of seven and so was not able to meet all the criteria for this study (case G-8).

Of the twenty-seven students in the clinic groups sixteen were off-spring of an affected and non-affected parent (group 1-A). A bias was created by the method used to sample family data, where one member (the proband) was affected with the disorder. Therefore, to analyze the data on a genetic basis, Weinberg's proband method was used to statistically adjust the data. All families with only one child were omitted from this analysis, since these children

do not have siblings with which to compare. This method assumes that the affected parent is a hybrid (heterozygous) and therefore, carries the gene for the disorder. When an affected and non-affected parent have children it is assumed that half of their children will be affected, and half will not. That is, theoretically, 50 percent of their children are expected to have this disorder.

With a large sample of progeny from a controlled experimental mating you could achieve a very close approximation of 50 percent being affected. Mendel in his original research with pea plants was able to achieve his ratio because of his large sample size, his ability to control the mating of the plants, and his method of measuring or observing the genetic characteristic was a simple one. In this investigation of specific reading disability, there was a small number of families, each having few family members, therefore, a small number of normal versus affected individuals to compare. Because of this, a larger deviation from the (theoretical) percentage could be expected if the reporting of any one family was inaccurate.

Table 7 shows a survey of the results of the Mendelian Analysis for group 1-A (affected and non-affected parent). The results were made without regard to the sex of the child. The Glassboro Clinic group obtained a Mendelian ratio of 37.7 percent with a standard error of ±9.9 percent. The University of Pennsylvania Clinic group obtained a Mendelian ratio of 31.3 percent with a standard

error of ±11.6 percent. When the two clinic groups were combined the Mendelian ratio was 35 percent with a standard error of ±7.5 percent. The control population obtained a Mendelian ratio of 7.7 percent with a standard error of ±4.3 percent.

Table 7

Survey of the Results of the Mendelian Analysis for Group 1-A. Parents Mating Affected X Non-affected Using Weinberg's Proband Method

Method	Formula	Glassboro Clinic	U. of Penn. Clinic	Control
No. of Families	ΣF	13	3	25
Families One Child		1	0	4
No. over 7 Years	Σs_i	37	19	64
No.affected Children	Σx_i	22	8	3
No. of Proband	Σy_i	13	3	0
No. Sibs of Proband	$\Sigma y_i(s_i-1)$	24	16	39
No.affected Siblings	$\Sigma y_i(x_i-1)$	9	5	3
Mendelian Ratio	$100\frac{\Sigma y_i(x_i-1)}{\Sigma y_i(s_i-1)}=P$	37.7%	31.3%	7.7%
Standard Error	$\sqrt{\frac{P(100-P)}{\Sigma s_i - F}}$	±9.9%	±11.6%	±4.27%
		Combined Clinics		
Mendelian Ratio		35%		
Standard Error		± 7.5%		

Chi square tests were performed to determine if these observed frequencies were significantly different from the expected 50 percent. The data in Table 8 is arranged in four columns, one each for the Glassboro Clinic Group, the University of Pennsylvania Clinic Group, the Combined Clinic Groups and the Control Students. For each of these groups, this table shows the number of siblings, the observed number of affected siblings, and the number of these siblings that

Table 8

Observed and Expected Frequencies, and Chi Square Values for Affected Siblings in the Glassboro Clinic Group, University of Pennsylvania Clinic Group, Combined Clinic Groups and Control Students

	Glassboro Clinic Group	U. of Penn. Clinic Group	Combined Clinic Groups	Control Students
Siblings	24	16	40	39
Expected Affected Siblings (50%)	12	8	20	19.5
Actual Affected Siblings	9	5	14	3
χ^2	0.75	1.125	1.80	13.96
	Not Sig.*	Not Sig.*	Not Sig.*	$p < .01$

*Not Significant: the chi square values obtained for the Glassboro Clinic Group, University of Pennsylvania Clinic Group and the Combined Clinic Groups indicate that there is no significant difference between the values obtained for these groups by the Weinberg proband method and the expected 50 percent in a group having a single hybrid dominant gene mode of inheritance.

could be expected to inherit the disorder by a single autosomal dominant gene.

Table 8 shows that the percent of siblings affected with specific reading disability in the clinic groups is not significantly different from the expected frequency of 50 percent. Thus it appears with a high degree of probability that specific reading disability is inherited following a single autosomal dominant gene mode of inheritance.

In group O-A and group C-A, the parents are identified as not affected by a reading disability. There were four students in group O-A and six in group C-A. It was not possible to do a genetic analysis on these students from such a limited amount of information. It should be noted that in three of these families, there was an indication that one of the parents had a reading disability, but not enough symptoms were noted to identify that parent as having a reading disability. (Cases G-7, G-12, G-17)

ANALYSIS OF OTHER DATA THAT MAY RELATE TO READING THAT WAS OBTAINED FROM THE FAMILY CHECK LIST

Education Level achieved by the Parents

Parents were asked to indicate the highest grade completed in school and at what age they began to read for pleasure. Table 9 shows a comparison of the educational levels of the fathers in the control and clinic groups. There appears to be no difference between the two groups.

The number of fathers that reported they never read for pleasure was three in the control group and eight in the clinic group. Of these fathers, one of the control group and six of the clinic group also had a reading disability.

Table 9

Education Levels Achieved by the Fathers and Mothers in the Clinic and Control Groups And Comparison of the Parents Who Do Not Like to Read for Pleasure

	Fathers				Mothers			
	Control (25)		Clinic (26)		Control (25)		Clinic (26)	
College Grad. or Higher		8		4		2		2
Partial College		3		0		4	(1N)	3
High School	(2N)	8	(3N)	9	(2N)	16		12
Less than High School	(1N)	6	(1N)	7	(1N)	2	(3N)	4
Total	(3N)	25	(4N)	20	(3N)	24	(4N)	21

(N) - Never read for pleasure.

Table 9 also shows the comparison of the educational levels of the mothers in the control and clinic groups. There appeared to be no difference between the educational levels obtained by the mothers of the control and clinic groups. There were three control mothers and four clinic mothers that reported never reading for pleasure. There appeared to be no difference in the enjoyment of reading between the two groups.

Biochemical Disorders That May Act as a Contributing Factor

The response to the biochemical factors that may have an effect on reading achievement are summarized by family and by student in the control and clinic groups in Table 10. Diabetes and low blood sugar (hyper- and hypo--glycemia) were tabulated together, as both are a malfunction of the pancreas and can produce similar effects on the body. There appeared to be no difference between the control and clinic groups in the number of families reporting this malady. No student in either group responded positively to this question.

Table 10

Summary of Biochemical Factors that may Influence Specific Reading Disabilities Comparing Control and Clinic Families, the Control and Clinic Students

Factors	Families		Students	
	Control (25)	Clinic (27)	Control (25)	Clinic (27)
Diabetes	10	13	0	0
Thyroid	15	16	0	2
Allergy	13	19	1	8

Both high (hyper) and low (hypo) thyroid problems were also tabulated together, as both affect the metabolism of the body. When the control and clinic groups were compared there appeared to be no difference between the fam-

ilies reporting this problem. Two clinic students reported thyroid problems conpared to no control students reporting this problem.

The frequency of allergy that was reported in the families of the control and clinic groups appeared to be not different. There was one student in the control group compared to eight students in the clinic population reporting allergy. These numbers are too small to draw any conclusions, but note should be taken of this.

Handedness and Mixed Dominance

Table 11 summarizes the number of individuals in the control families (114) and the number of individuals in the

Table 11

Summary of Responses of Family Members Being Right Handed, Left Handed, Ambidextrous and Mixed Dominance

	Control (25 Families)				Clinic (27 Families)			
	Stud.	Sibs.	Parent	Total	Stud.	Sibs.	Parent	Total
Right Handed	21	34	47	106	23	54	53	130
Left Handed	3	4	3	10	4	8	1	13
Ambidex.	0	0	0	0	0	0	0	0
Total	25	39	52	114	27	62	54	143
Mixed Dominance Noted in Each Group	3	9	8	20	2	8	7	17

clinic families (143) that reported being right handed, left handed or ambidextrous. Also the number of individuals who were reported as being mixed dominant (mixed hand and eye preference) was reported. When the number of persons in the control and clinic groups were compared, the results appeared to show no differences in the two populations.

SUMMARY

A clinical and genetic analysis was made on the basis of fifty-two families; twenty-seven were from reading clinics where at least one child in the family had been diagnosed as having a reading disability. Twenty-five were control families. A summary was made of specific reading disability characteristics found in the families, and the students used in this study. There was a larger number of males than females affected with a reading disability in this study. Birth order does not appear to be a contributing factor in this study.

Only one family was present where both parents had a reading disability; both of their children also were affected with a reading disability. In the families where one parent was affected with a reading disability, Weinberg's proband method of Mendelian Analysis was used, giving a 37.7 $\pm$9.9% of their children being affected. The results of the chi square test performed, indicated that the value of 37.7 percent is not significantly different from the theoretical 50 percent expected in a single hybrid auto-

somal dominant mode of inheritance. Therefore, the null hypothesis was rejected, as there is a relationship between heredity and specific reading disability.

Chapter 5

SUMMARY, CONCLUSIONS, AND IMPLICATIONS

SUMMARY

Reading disabilities, unless associated with obvious neurological damage, have come to be regarded as psychological or pedagogical problems. It was the purpose of this study to investigate the relationship between heredity and specific reading disability. The part that heredity plays in reading problems has received the least amount of investigation of all the recognized possible causes for reading disability.

There are many family case studies and descriptive reports in the literature that tell of several members of a family being affected by a reading disability. These date back to the turn of the century. Hallgren's (1950) clinical and genetic study is the most complete study available.

There is much inconsistency in the naming and defining of this reading difficulty. In this thesis, specific reading disability was defined operationally as a failure to learn to read with normal proficiency despite conventional instruction, a culturally adequate home, proper motivation, intact senses, normal or above normal intelligence and freedom from gross neurological defects.

As a result of the review of the literature, a questionnaire, called the Family Check List, was developed to obtain the needed family background information relevant to genetic characteristics and reading disabilities.

The Family Check List was mailed to three groups; a control group, a Glassboro Reading Clinic group, and a University of Pennsylvania Reading Clinic group. There were a total of twenty-eight families in the clinic groups and twenty-five families in the control group, giving a total of 257 persons studied. The information received on each individual was analyzed to see if enough characteristic symptoms were present to determine if that person could be classified as having specific reading disability. A summary was made of specific reading disability characteristics found in the families and the students used in this study.

The families were divided into four groups for the purpose of clinical analysis; (1) families with both parents affected with a reading disability, (2) families with only one parent affected with a reading disability, (3) families where the proband and at least one sibling and/or grandparent were affected with a reading disability but not the parents, (4) families where just the proband was affected.

In the distribution of students with specific reading disability, more males than females were affected. When the affected sibling and parents were tallied and added to the number of affected students, the male to female ratio remained the same. The position of the

affected children in the birth series does not appear to be a contributing factor in this study.

There was only one family with both parents having a reading disability; both of their children also were affected with a reading disability. In the families where cnly one parent was affected with a reading disability, Weinberg's proband method of Mendelian Analysis was used, giving a 37.7 $\pm$9.9 percent of these children being affected. The results of the chi square test performed indicated that the value of 37.7 percent was not significantly different from the theoretical 50 percent expected. In the remaining groups, the parents could not be identified as having specific reading disability and it was not possible to do a genetic analysis from such a limited amount of information for these families.

This study did not appear to show any difference between the clinic and control groups in reporting hyper- or hypo-glycemia (blood sugar) or thyroid problems. But, there were more students in the clinic groups reporting allergies. These numbers were too small to permit drawing conclusions; but note should be taken of this. When the number of individuals in the clinic and control groups were compared as being right handed, left handed, and mixed dominant, the results appeared to show no difference in the two populations.

CONCLUSIONS

Validity of the Check List

Fifty-two families filled out and returned the Family Check List used in this study. The characteristic symptoms of specific reading disability used in this study were present in both the control and clinic families in varying numbers. This agrees with Herman and Rabinovitch, who both state that these characteristics are found in many readers, but they are more pronounced and remain with the disabled reader for a longer period of time, sometimes into adult life.[1, 2] When the control and clinic groups of students were compared, there appeared to be a difference in the number of times each characteristic was reported for the clinic groups.

When the parents were given the questions on the check list as a guide, they could recall if the characteristic symptoms were present in their family. Many wrote notes about the answers they gave. The parents' responses about their children given on the check list were checked against the clinical reports in the children's records (when available). This seemed to indicate that parents could reply accurately to the characteristic symptoms of

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[1]Knud Herman, "Specific Reading Disability," Danish Medical Bulletin, II, 1 (1964), 35.

[2]Ralph Rabinovitch, "Reading and Learning Disability abilities," American Handbook of Psychiatry (New York: Basic Books, 1959), p. 867.

specific reading disability they had noted in their child or children. The parents could recall if reading, spelling or writing was a problem for them, but some could not recall the degree of their own reading difficulty or type of errors they used to make. Others wrote notes as to the type of reading difficulties they had or still encountered. Three families were called by the researcher after the check list was returned, to check on the age of a child, or which brother or sister had certain characteristics indicated on the check list. This experience indicates that if the check list could be mailed to the family and after completion used as the basis for part of a family interview, a very good familial picture could be expected.

The questions on the Family Check List which received few responses were number thirteen, difficulty with a foreign language, and number twenty-four, right and left confusion. Eight clinic families compared to four control families responded positively to question thirteen; only one clinic student and no controls responded positively to this question. This was due in part to the fact that many of these students have not studied a foreign language. Right and left confusion is a characteristic symptom of specific reading disability which is reported very frequently in the literature. Either this question was not worded properly, or the families in this study were unaware of the difficulty they were having in right and left confusion. They may have felt that this was the common experience for most people.

Not all families reported on reading disabilities in grandparents, therefore the data gathered for them, and other relatives could only be used to clarify the reading disability in that family. These data were not used in the tabulation in this thesis. The Family Check List appeared to be able to differentiate families in which there is a tendency toward reading disabilities, but the Family Check List has not yet been tested for reliability.

Heredity and Reading Disability

The result of the Mendelian Analysis computed on the data in this study seems to indicate that specific reading disability is inherited. The following are the findings that have led to this conclusion.

Position of affected child in birth series. If a disorder is inherited, the distribution of the affected members in the birth series will be random. If it is significantly non-random, for example, if the first-born had specific reading disability more frequently than other positions in the birth series, the null-hypothesis would have to be accepted. In this study, the birth order does not appear to be a contributing factor in these cases of specific reading disability. The results of this analysis therefore support the hypothesis, that specific reading disability is a hereditary disorder.

Mode of inheritance. The possibility of the reading

disability having a recessive sex-linked mode of inheritance on the X-chromosome is ruled out because in families with both sons and daughters affected, the father was not always affected. Specific reading disability occurs in both sexes, and in this study it was transmitted in some families from father to son and daughter, and in other families from mother to son and daughter. Therefore, the Y-chromosome linkage can be ruled out, as only males are affected with Y-chromosome characteristics.

In the distribution of the affected students in the clinic population, the males outnumbered the females. When the affected siblings and the affected parents were also tallied, the ratio of males to females remained the same. The higher incidence of males may be due to sex-influenced inheritance, because there are more affected males than females. Sex-influenced genes are not located on the sex chromosomes, but are a developmental expression of the gene in one sex more than the other (as baldness is more common in men than in women). Most sex-influenced genes are autosomal (not on a sex chromosome), are carried by male and female, and simple inspection can show their sex-influencing effect. This study seems to varify that specific reading disability is inherited by a single gene on an autosomal chromosome.

The analysis of the families in this study strongly supports the hypothesis that specific reading disability follows a dominant mode of inheritance. When a gene is

dominant, only one gene is needed and it can come from either parent. In group 2-A, there was only one family; both parents were affected and both of their children had been diagnosed as having a reading disability. If both of the parents were heterogygous (Dd), then three out of four of their children, 75 percent should also be affected. If one of the parents is homozygous (DD), the expectation is 100 percent. Group 2-A was too small for a Mendelian ratio determination.

In Group 1-A only one of the parents is affected with specific reading disability. When the mode of inheritance is monohybrid dominant (only one parent has the dominant gene for the disorder) the theoretical expectation for the Mendelian ratio is 50 percent. Using Weinberg's proband method to statistically adjust the data and remove sampling bias, a Mendelian ratio of 37.7 percent $\pm$9.9 percent was obtained for the Glassboro Clinic Group and 31.3 percent $\pm$11.6 percent for the smaller University of Pennsylvania Clinic Group. Using chi square, these findings for the Clinic groups were not significantly different from the theoretical 50 percent expected. The Mendelian analysis of the families in this group show, with a high degree of probability, that specific reading disability follows a single autosomal dominant gene mode of inheritance.

The occurrence of a hereditary disorder in three successive generations, argues in favor of a dominant mode of inheritance. The information obtained about the grand-

parents was not adequate to make it possible, in all cases, to determine whether grandparents had a reading disability or whether their difficulty was due to foreign birth or lack of education. Therefore, no conclusions could be drawn from this information to either aid in the accepting or rejecting of the null-hypothesis.

In group O-A and C-A, the parents could not be identified as having specific reading disability. They did not have a reading disability, not enough information was given to so classify them, or they only filled out the Check List with information pertaining to the student. It was not possible to do a genetic analysis on these groups with such a limited amount of information. In three of these families there was an indication that one of the parents had a reading disability, but not enough symptoms were noted to identify that parent as affected with the disability.

The other possibility is that when only one person in a family is affected with the disability for several generations, this could be a reading disorder based only on environmental causes. It is not possible to differentiate between hereditary and environmental reading disabilities on the basis of symptoms only.

Biochemical factors. Variations in the biochemical balance of the body may also affect children with specific reading disability, causing changes from day to day in their ability to learn. If a child's biochemical state is in

balance, and he is not suffering from allergies, his ability to learn should be more consistent. A similar phenomenon is characteristic of hemophilia; that is, if a hemophiliac is in perfect health and is bruised he may not "bleed" at all, but if he has a cold, or any biochemical imbalance, and bruises himself he will bleed and need medical attention.

This study did appear to show no difference between the clinic and control groups in reporting of hyper- or hypo--glycemia (bood sugar) or thyroid probl ems. There were more students in the clinic groups reporting allergies. The numbers were too small to form any conclusions.

The infcrmation used in this study was obtained from family background. To investigate this further a medical and biochemical approach would be advisable.

Environmental effects on the gene. The degree of severity of the reading disability and its characteristics vary from child to child. This may be because of the nature of the gene. The genetic constitution of a gene is termed genotype. The genotype of a person remains constant, fixed at the time of conception. The external appearance of a gene is termed phenotype. The phenotype is potentially variable, the result of the interaction between genotype and its non-genetic environment. Specific reading disability is a phenotypic expression of this gene.

If a child is born carrying the gene for specific reading disability, and all his environmental conditions,

pre- and post-natal, are optimal, the child may be only slightly affected by the disability. But, if a child carrying this gene has some adverse environmental condition, either pre- or post-natal, the degree of reading disorder will be greater. This might account for the fact, that lack of oxygen at birth, falling on the head, and other physiological misadventures appear to result in reading disabilities in some children but not in others.

The environmental influence on the gene may be from an internal or external cause. Such things as biochemical imbalance, a difficult birth, high fever, can all be classed as internal. The external environment can also affect the phenotype expression of the gene. Because of the child's disability, some learn better by one method than others, but the optimum method will vary from child to child. Some learn better through auditory channels, some through visual channels, some through tactile channels, and some learn better through a particular combination of channels. If a child is diagnosed as having a reading disability early, and taught to read by the method that is best suited to his strengths and weaknesses, his reading disability will be much less. Therefore, the child's hereditary background, the method by which he is educated, and his environment are all responsible for his reading disability.

IMPLICATIONS

If the hypothesis that specific reading disability is

inherited were accepted, many unanswered questions about specific reading disability would be answered. This hypothesis would account for the variations in the severity of the reading problem and the different characteristic symptoms noted from child to child. It would account for the fact that reading disability of children in the same family is not exactly the same. It would account for the fact that some children who presumably do not carry the gene can experience the same environmental mishaps as the child carrying the gene, and not have a reading disability with the characteristic outward signs. This would account for the success of the predicting reading failure type of questionnaire that obtains the child's pre-school information.

If we are aware of the underlying cause of specific reading disability, teachers should then know how to teach these children to overcome their difficulty. Too often the outward signs are given as the reason for a child not achieving, instead of using these signs as a guide in helping the child. Some writers feel that specific reading disability does not exist, because if a child is properly taught from the beginning, by the method that is best suited to him, he is able to overcome the disability[3] to the extent that many of the phenotypic characteristics of this gene can be overcome and he can learn to read.

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[3]Morton Botel, "Methods and Systems for Teaching Dyslexic Pupils," Dyslexia: Diagnosis and Treatment of Reading Disorders, ed. A. Keeney (St. Louis: Mosby Co., 1968), pp. 120-30.

Therefore, the responsibility for this child rests with: (1) The mother, who needs to have adequate pre-natal and post-natal care for her children in order to avoid any internal environmental mishaps. (2) The mother, who needs to know more about how her preschool child learns and how she can help him in addition to satisfying his other needs. (3) Early childhood education programs which may need more perceptual type programs integrating all phases of perceptual training, not just one. (4) The teacher, who needs to be the best informed, most experienced, perceptually aware teacher with a good knowledge of reading. These teachers are needed most in the early grades and should be able to teach to each child's strengths and weaknesses, by the method that is best suited for the child.

RECOMMENDATIONS FOR FURTHER RESEARCH

1. The Family Check List must be further tested for reliability and validity. The reliability of the Family Check List could be checked by first having it filled out at home, then using it as part of the interview procedure to verify the familial background.
2. The null-hypothesis of this study should be investigated on a larger sample population.
3. The null-hypothesis of this study should be investigated by having siblings and parents, also, completely diagnosed for reading disabilities, using the WISC, reading

tests, spelling test, etc.

4. A genetic study of reading disabilities should be done from a genetic-medical research point of view. The mystery of the gene and chromosome is now beginning to be unraveled and a study of the type and placement of this gene could be of much value.
5. The sex distribution of specific reading disability should be investigated, especially in light of the ratio of males to females in the families in which the disability is found. (Normal population ratio is 1:1 males to females.) There was a 2:1 ratio of males to females when the proband was counted, and 3:2 when he was not counted.
6. The effect of biochemical imbalances, and allergies in the children and their families with specific reading disability should be investigated to find out if they are contributing factors.
7. The effect of different methods of teaching on the child with a reading disability should be investigated, and this information passed on to all classroom teachers.

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APPENDIX A.

Diagnostic & Consultation Center
Glassboro State College
Glassboro, New Jersey 08028
January 5, 1971

Dear Parents:

May I ask your assistance as I pursue original research in reading at Glassboro State College. Many children have difficulty in learning to read. Although much research is being done to discover why this is so, the problems of children who cannot read are still with us.

You can help us. Information is needed about how people in a family read. Enclosed, you will find a Family Check List. The information from the checklist may help us in discovering some of the reasons why certain children, and adults have difficulty in reading and others do not. I am gathering information from all types of readers.

Please answer the questions as best you can. If you are not sure about how to answer a question, just write what you think about it in the comment area. If you have additional information that you feel may be helpful in my research, please write this on the back of the papers.

In addition to the checklist, have the student draw a picture of a man and write a sentence about it. Please do not give the child any assistance with the drawing or spelling.

It would be greatly appreciated if you would complete the Family Check List as soon as possible and return it in the enclosed envelope.

Thank you very much for your cooperation.

Sincerely yours,

Laryl Lee Delker

Laryl Lee Delker
Graduate Assistant

Bells Elementary School

WHERE EACH CHILD COUNTS

GREEN TREE ROAD BLACKWOOD, N. J. 08012

HOWARD S. HAUSMANN
PRINCIPAL

TELEPHONE
(609) 589-5500

January 12, 1971

Dear Parents:

By means of this letter I should like to introduce Mrs. Laryl Lee Delker. Mrs. Delker is a full time graduate assistant working for a Master of Arts degree at Glassboro State College.

I have worked and studied with Mrs. Delker and know her to be a sincere and dedicated person very much interested in helping boys and girls with reading problems.

Mrs. Delker has designed a questionnaire to be used as a tool for gathering information. The questionnaire is to be tested on a group of students having specific reading disability, a group of students having reading problems, and a control group of average and better readers.

Your child has been selected from the group who are average or better readers. I'm proud that this group of better readers were selected at the Bells School and that we will have a part in Mrs. Delker's research.

You and your child can be a part too by carefully following the instructions enclosed and forwarding this confidential information to Mrs. Delker at the college. Your child will not be identified beyond his first name.

Please accept my appreciation for your assistance in this study.

Very sincerely,

Howard S. Hausmann

HSH/mr

Family Check List

Laryl Lee Delker Form

1. Student's first name _____
2. Date of birth _____ Age ___ Sex ___
   month day year
3. Grade in school _____
4. Father's occupation _____
   A. Highest grade completed in school _____
   B. When did he begin to read for pleasure? (give approximate age, or never) _____
5. Mother's occupation _____
   A. If answer is housewife, what type of work did she do before marriage? _____
   B. Highest grade completed in school _____
   C. When did she begin to read for pleasure? (give approximate age, or never) _____
6. Brothers and sisters

	First Name	Sex	Age	Any difficulties in school? What subjects?
1.				
2.				
3.				
4.				
5.				
6.				

Person who completed form:

_____

Date _____

Instructions: For the following questions, please circle the family member or members to whom the questions would apply. Add any additional comments in the third column and on the back of the paper.

Student refers to the name on line 1.

Sister(s) and Brother(s) of student. If circled, give the name of that person in the comment column.

Others refers to Uncles, Aunts, and Cousins. If circled, give the relationship to the child in the third column.

Question	Circle the member of the family to whom the answer applies.	Additional Comments
7. Are any members of the family identical twins?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
8. Are any members of the family adopted?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
9. Have any members of the family experienced difficulty with reading?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
10. Are any members of the family now having difficulty with reading?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
11. Do any members of the family experience difficulty with spelling?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
12. Do any members of the family have poor handwriting for their age?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
13. Have any members of the family experienced unusual difficulty in learning a foreign language in school?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
14. Do any family members understand many more words in conversation than they can read?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
15. Have any members of the family ever had a speech problem? (Example: stuttering, lisp, difficulty saying certain letters)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
16. Have any members of the family ever confused or reversed letters when reading or writing? (Example: b and d; on and no; was and saw)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
17. Have any members of the family ever confused sounds of letters? (Example: d and t; v and f; vowels in words like pin and pen)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
18. Have any members of the family had difficulty with reversing numbers? (Example: 21 for 12; 78 for 87)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
19. Have any members of the family had difficulty reading musical notes, math symbols, shorthand or Morse Code?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
20. Do any members of the family have difficulty remembering numbers? (Example: frequently recheck telephone number while dialing)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
21. Have any members of the family had trouble using a dictionary or telephone book because they are unsure of location of letters in the alphabet?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
22. Do any members of the family read very slowly?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
23. Do any members of the family substitute or omit words when they are reading?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
24. Do any members of the family have trouble telling left and right in following directions?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
25. Do any members of the family have diabetes?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
26. Do any members of the family have a low blood sugar (get very tired, cross and sometimes hungry before mealtime?)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
27. Have any members of the family ever been told by a doctor that they should take thyroid pills?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
28. Are any members of the family tired a great deal; have cold hands and feet when in a heated building?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	
29. Has any member of the family ever been told by a doctor that he has an over-active thyroid?	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

Question	Circle the member of the family to whom the answer applies	Additional Comments
30. Do any members of the family have allergies? (Specify what kind in the comment column)	Student Sister(s) Brother(s) Mother Father Mother's Mother Mother's Father Father's Mother Father's Father Others	

In the following questions, place after each of the family members listed an R if right, L if left, E if can use either right or left equally well. If unsure of answer, leave blank.

31. Which hand do they use for writing?	Student ___ Mother ___ Father ___ Mother's Mother ___ Mother's Father ___ Father's Mother ___ Father's Father ___ Sister(s) ___ Brother(s) ___	

32.

Which foot do they use to kick a ball?

Student ___
Mother ___ Father ___
Mother's Mother ___
Mother's Father ___
Father's Mother ___
Father's Father ___
Sister(s) ___
Brother(s) ___

33.

Which eye would they use to look through a small hole?

Student ___
Mother ___ Father ___
Mother's Mother ___
Mother's Father ___
Father's Mother ___
Father's Father ___
Sister(s) ___
Brother(s) ___

34. On this paper, have the student draw the best picture of a man that he can.

Have student write a sentence about the picture.

_____

_____

_____

First Name _____ Age _____

APPENDIX B.

FAMILY CHECK LIST - Tabulation Sheet

Child's name _____ No. ____ Age ____ Sex __

Adopted ____ Twin ____ Draw-A-Man ____ I.Q. __

	Child	Mother	Father	Mat. Gr.M.	Mat. Gr.F.	Pat. Gr.M.	Pat. Gr.F.	Sisters				Brothers				Others
9. Had rdng diff.																
10. Has rdng diff.																
11. Spell. diff.																
12. Poor handwriting																
13. Foreign lang.																
14. Larger spk voc.																
15. Speech problem																
16. Reverse letters																
17. Snds of letters																
18. Reverse letters																
19. Other codes																
20. Digit span																
21. Alphbt order																
22. Very slow rdr																
23. Omit words																
24. L/R direction																
25. Diabetes																
26. Low blood sugar																
27/8. Low thyroid																
29. High thyroid																
30. Allergy																
31. Hand																
32. Foot																
33. Eye																

SUMMARY OF DATA

Selected information about the fifty-two families obtained from the Family Check List is given in the following tables. The student, his siblings and parents are summarized: (M=Male, F=Female, G=Glassboro Reading Clinic, P=University of Pennsylvania Reading Clinic, C=Controls). If there is a (+) in the reading disability columns, this indicates that person not only responded positively to question 9 or 10 (has a reading difficulty) but also, characteristic symptoms of specific reading disability were indicated on the Family Check List. (A) means affected with a reading disability. If given information was not enough to clarify the type of reading disability a (?) was used.

SUMMARY OF CLINIC FAMILIES WITH
SPECIFIC READING DISABILITY

Case	Children >7 yrs	Student Sex	Student Disability R	Student Disability S	Student Disability A	Affected Siblings Sex	Affected Siblings Disability R	Affected Siblings Disability S	Affected Siblings Disability A	Father Disability R	Father Disability S	Father Disability A	Mother Disability R	Mother Disability S	Mother Disability A
Group 2-A															
G-8	2	M	+	+	A	M	+	+	A	+	+	A	+	+	A
Group 1-A															
G-3	2	M	+	+	A					+	+	A			
G-4	3	M	+	+	A	M	+	+	A				+	+	A
G-5	2	M	+	+	A	M	+	+	A	+	+	A			
G-6	1	F	+	+	A					+	+	A			
G-10	3	M	+	+	A	M	+	+	A				+	+	A
G-11	2	M	+	+	A	M	+	+	A				+	+	A
G-13	3	M	+	+	A	M	+	+	A	+	+	A			
G-14	4	F	+	+	A	M	+	+	A	+	+	A		+	
G-15	2	F	+	+	A	M	+	+	A				+	+	A
G-16	6	M	+	+	A					+	+	A			
G-18	4	M	+	+	A	M	+	+	A				+	+	A
G-21	2	M	+	+	A					+	+	A			
G-25	3	M	+	+	A	M	+	+	A	+	+	A			
P-1	5	M	+	+	A	M/M	+	+	A/A	+	+	A	+	?	

SUMMARY OF CLINIC FAMILIES WITH
SPECIFIC READING DISABILITY
(Continued)

Case	Children >7 yrs	Student Sex	Student Disability R	Student Disability S	Student Disability	Affected Siblings Sex	Affected Siblings Disability R	Affected Siblings Disability S	Affected Siblings Disability	Father Disability R	Father Disability S	Father Disability	Mother Disability R	Mother Disability S	Mother Disability
P-3	8	M	+	+	A	M/M	+	+	A/A				+	+	A
P-4	6	M	+	+	A	M	+	+	A	+	+	A			
Group O-A															
G-2	2	M	+	+	A	F	+	+	A				?	+	?
G-12	5	M	+	+	A	M/F	+	+	A/A	?	+	?			
G-17	3	M	+	+	A	M	+	+	A	?	+	?	?	+	?
G-22	5	F	+	+	A	M	+	+	A						
Group C-A															
G-1	2	M	+	+	A										
G-7	3	M	+	+	A			+	?		+				
G-9	3	M	+	+	A										
G-20	2	M	+	+	A										
G-26	5	M	+	+	A										
G-27	2	M	–	+	A										

SUMMARY OF CONTROL FAMILIES WITH
READING DISABILITY

Case	Children >7 yrs	Student Sex	Student Disability R	Student Disability S	Affected Siblings Sex	Affected Siblings Disability R	Affected Siblings Disability S	Father Disability R	Father Disability S	Mother Disability R	Mother Disability S
C-1	1	M									
C-2	2	M						+		+	
C-3	2	M									+
C-4	2	M									+
C-5	2	F									
C-6	2	F									
C-7	1	F						+		+	
C-8	3	F									
C-10	2	F									
C-11	2	F								+	+ A
C-12	2	M									
C-13	6	M									
C-14	3	M									
C-16	2	F			M	+	+ A				
C-17	3	F									
C-19	1	F									
C-21	3	M						+			

SUMMARY OF CONTROL FAMILIES WITH
READING DISABILITY
(Continued)

Case	Children > 7 yrs	Student Sex	Student Disability R	Student Disability S	Affected Siblings Sex	Affected Siblings Disability R	Affected Siblings Disability S	Affected Siblings	Father Disability R	Father Disability S	Father	Mother Disability R	Mother Disability S
C-22	1	M											
C-23	3	M			M	+	+	A		+			
C-24	5	F											
C-25	3	F											
C-26	2	F											
C-27	4	F			F	+	+	A	+	+	A		
C-28	4	F											
C-29	3	F											